enrolled into the ACOS-Registry (Acute Coronary Syndrome, 154 hospitals) in Germany. We analysed the prospective data of STEMI-pts with special respect to the smoking status.

Results: Out of 5122 consecutive pts with STEMI, 1852 (36%) were current smokers and 2753 (54%) had never smoked (nonsmokers). Smokers were at mean 15 years younger, more often male and had less often hypertension and diabetes as additional cardiovascular risk factors. Smokers did receive acute reperfusion therapy more often. The hospital mortality in nonsmokers was three times higher than in smokers. After correcting for differences in baseline characteristics and acute reperfusion and adjunctive therapy in a multivariate analysis smoking did not influence hospital mortality after STEMI (OR 0.80, 95% CI 0.60-1.06).

Conclusion: In smokers STEMI occurred 15 years earlier in age than in nonsmokers. In a multivariate analysis smoking did not influence the outcome of STEMI.

Parameter	Smokers n=1852	Nonsmokers n=2753	p-value
Age (years)	56	71	<0.01
Male gender	81.6 %	60.4 %	<0.01
Prior MI	8.4 %	12.8 %	<0.01
Hypercholesteremia	47.4 %	40.2 %	<0.01
Hypertension	45.0 %	63.3 %	<0.01
Diabetes	16.1 %	30.5 %	<0.01
CK max (U/l)	625	532	<0.01
Acute Reperfusion	78.7 %	64.8 %	<0.01
Thrombolysis	26.7 %	20.7 %	<0.01
Primary PCI	52.0 %	44.1 %	<0.01
Hospital Mortality	4.7 %	13.3 %	<0.01

8:45 a.m.

869-2 Comparison of the Importance of Increasing Pathogen Burden, Elevated C-Reactive Protein, and the Presence of Antibodies to Heat Shock Protein 60 on Myocardial Infarction or Death

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In addition to the importance of traditional risk factors, recent evidence suggests that infectious pathogens, C-reactive protein (CRP) and heat shock protein (HSP) play roles in the progression of atherosclerosis and can be used as outcome predictors. In the present study, we compared the relative importance of a) pathogen burden (seropositivity to cytomegalovirus, hepatitis A virus, C. pneumoniae, H. pylori, herpes simplex virus type 1 and type 2), b) CRP levels, and c) anti-human HSP60 antibodies in predicting myocardial infarction (MI) or death. The patient cohort consisted of 890 patients (77% men, mean age 65 years) with coronary artery disease (CAD) documented by coronary angiography ($\geq$ 70% stenosis). The mean follow-up period was 3 years. By both univariate and Cox multivariate regression analyses, pathogen burden and elevated CRP levels were strong and independent predictors of incident MI or death. The highest relative hazard was conveyed by pathogen burden (Table 1). In contrast, HSP60 antibodies were not significant determinants of MI or death. We conclude that in patients with documented CAD, both pathogen burden and elevated CRP levels are important independent predictors of MI or death, suggesting that infection and inflammation contribute to the disease processes leading to acute coronary occlusion. Although HSP60 has been associated with angiographic evidence of the presence and extent of CAD, it's role in the acute complications of CAD is still to be determined.

Table 1. Comparison of Relative Hazard (95%CL) of Incident MI or Death

	Unadjusted	Adjusted For CAD Risk Factors
Pathogen Burden0-3	1.0	1.0
4	1.9 (0.8-4.4)	1.5 (0.6-3.5)
5	3.2 (1.4-6.9)	2.6 (1.2-5.7)
6	4.6 (2.1-9.9)	3.1 (1.4-6.8)
CRP Tertile 1	1.0	1.0
2	1.9 (1.3-2.9)	1.8 (1.2-2.6)
3	1.9 (1.3-2.9)	1.7 (1.1-2.5)
HSP60 Ab (+)	1.0	1.0
HSP60 Ab (-)	1.2 (0.8-1.6)	1.2 (0.8-1.6)

9:00 a.m.

869-3 Increased Concentrations of Bone Marrow-Derived Stem Cells in Peripheral Blood After Acute Myocardial Infarction

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Background: recent studies in experimental models have shown that bone marrow-derived stem cells (BMSCs) can regenerate myocardial tissue after a myocardial infarction. Moreover, the presence of cardiomyocytes of extracardiac origin has been reported in human transplanted hearts. It is reasonable to assume, therefore, that BMSCs may be released into the peripheral blood after an acute myocardial infarction (AMI), migrate towards the heart, and there differentiate into cardiomyocytes to repair, at least in part, the damaged tissue. The aim of this study was to evaluate whether BMSC concentrations increase in peripheral blood after an AMI. **Methods:** BMSC concentrations were measured by flow-cytometry as CD34+ cells/microliter (mcl) in the peripheral blood of 22 patients with AMI after 1, 3, 5 and 7 days from the event and in 11 healthy controls without cardiovascular risk factors, of similar age, sex and race. **Results:** In agreement with the literature, the median concentration of CD34+ cells in healthy controls was <1/mcl (0.47/mcl, interquartile range - IR - 0 to 0.94). In contrast, in the absence of clinical and laboratory findings of hemoconcentration, the median concentration of CD34+ cells in patients with AMI was significantly increased at 1, 3, 5 and 7 days from the event to 4.46/mcl (IR 2.6 to 8.4, p=0.0005), 2.91/mcl (IR 2.16 to 4.045, p=0.0014), 5.03/mcl (IR 2.38 to 7.9, p=0.0013), and 7.925 (IR 5.26 to 12.68, p=0.0075), respectively (Figure). **Conclusions:** In patients with AMI there is an increase in peripheral bone marrow-derived stem cell concentrations. These results support the hypothesis that these cells may migrate, differentiate, proliferate and contribute to remodel the infarcted myocardium.

9:15 a.m.

869-4 Association of Left Ventricular Hypertrophy Regression and Heart Rate Reduction With Determinants of Myocardial Oxygen Consumption and Cardiovascular Events: The LIFE Study

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Background: Experimental studies have demonstrated that increased myocardial O2 consumption demand due to left ventricular hypertrophy (LVH) increases the likelihood and size of myocardial infarction (MI) with a standardized coronary occulusion, but no human data exist to separate the potentially beneficial effects of LVH regression and reduced myocardial O2 demand on the rate of MI in treated hypertensive patients. Methods: In a double-blind, randomized, parallel-group design 960 participants in the Losartan Intervention for Endpoint reduction in hypertension (LIFE) study (average age 66 years, blood pressure 174/98 mmHg) and ECG-documented LVH were assigned once daily losartan- or atenolol-based therapy and underwent echocardiography after 1,2,3,4 and 5 years' treatment. LV mass and the LV mass x end-systolic stress x heart rate triple product were measured as indices of LVH and O2 demand. Results: Blood pressure was reduced similarly in the two treatment arms (mean=-29.7/16.2 versus -28.0/16.0 mmHg, NS) but heart rate fell more on atenolol (mean=-7.2 versus -1.1 beats per minute, p<0.001). LV mass was reduced more in losartan than atenolol-treated patients (mean=-22.0 versus -17.7 g/m2, p=0.021). In contrast, the triple product fell more on atenolol-based therapy (mean=-28% versus -18%, p<0.001). In Cox regressions adjusting for treatment, baseline LV mass index and baseline and in-treatment pressures, each 25 g/m2 lower LV mass index was associated with risk reductions (95% CI) of 22 (5-34, p=0.02)% for stroke and 33 (17-43, p=0.001)% for cardiovascular (CV) mortality but only 13 (33 t0 -17, NS)% for MI. Conclusion: Losartan-based antihypertensive therapy reduced LV mass more but atenolol reduced the triple product (index of myocardial O2 demand) by 10% more, potentially contributing to less reduction in MI than stroke or CV death associated with reduction of LV mass and to less effect of losartan than atenolol on MI than stroke or CV death in the entire LIFE study.

9:30 a.m.

869-5 The Assessment of Myocardial Viability With Delayed Contrast Enhancement Using Computed Tomography

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Background: Delayed enhancement of contrast has emerged as the most popular MRI approach to detect ischemic injury to myocardium. We have developed a CT technique for the quantitative measurement of myocardial distribution volume (MDV) of contrast media using retrospective ECG gated cine scanning.

Methods: Four 15-25 kg beagles were used for the study. For MDV measurement, a 30 s baseline (without contrast) cine scan was performed in synchrony with ECG recording using a General Electric Medical Systems (GEMS) LightSpeed multi-slice CT scanner. Contrast (Omnipaque, 225 mgI/ml) was then constantly infused for 30-60 min before the scan was repeated. Images were reconstructed with half-scan data (330 ms) at 0.1 s interval and those at end-diastole (ED) were selected with SmartScore (GEMS) and averaged with CT Perfusion 2 (GEMS). MDV maps were generated by subtracting the averaged non-contrast enhanced ED images from the corresponding averaged constant infusion images. The difference images were normalized to the enhancement in the